FACTORS DETERMINING SHORT TERM GRAFT FUNCTION IN RENAL TRANSPLANT RECIPIENTS

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THE TAMIL NADU
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CERTIFICATE

This is to certify that this dissertation entitled FACTORS DETERMINING SHORT TERM GRAFT FUNCTION IN RENAL TRANSPLANT RECIPIENTS submitted by Dr. S.Jayalakshmi, appearing for the D.M., Nephrology Degree Examination in August 2008 is a bonafide record of work done by her under my direct audience and supervision in partial fulfillment of regulations of the Tamilnadu Dr. M.G.R. Medical University, Chennai, Tamilnadu, India.

Prof.Dr.M.JAYAKUMAR, M.D.,D.M.,
Professor and Head,
Department of Nephrology,

Madras Medical College and
Research Institute,
Government General Hospital
Chennai - 600 003.

Dean,
Madras Medical College
and
Research Institute
Government General Hospital
Chennai - 600 003

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CONTENTS

INTRODUCTION

The first successful kidney transplants in humans were from identical twin living donors. Although transplanted before the development of chemical immunosuppression, many of these identical twin grafts had long-term survival. With recognition of the immunosuppressive effects of prednisone and azathioprine, the use of non twin donors became possible. Considerable controversy soon followed as to whether it was ethical to use living donors for kidney transplantation. Proponents of the use of living donors noted that the short and long-term patient and graft survival rates were better after living (vs. cadaver) donor transplants. Opponents worried that living donor nephrectomy was a major operation with potential risk to the donor; they believed that these risks did not justify the benefits to the recipient [1].

The biggest challenge in transplantation today is increasing the number of available organs. However, in the past 2 years, the number of living donor transplants in the United States has increased. Much of this increase followed the recognition that living unrelated donor recipients had out-comes similar to those of living related non-HLA-identical donor recipients [1].

A number of recent analyses demonstrate the importance of events early post-renal transplantation in determining long-term allograft outcomes. In particular, worse long-term outcomes are associated with delayed graft function (DGF), irrespective of the occurrence of acute rejection[2]. Attempts have therefore been made to improve early graft function by a variety of mechanical, pharmacological and organ allocation strategies. If suboptimal early graft function could be accurately predicated, the success of these strategies may be improved[3].

The serum creatinine at 1 year, rather that the serum creatinine at 3 or 6 months, was found from this analysis to be an excellent predictor of the long-term survival of the graft as was reported by Hariharan. It was also found to be an important factor in a recent report by He and Johnston. With this background, we wanted to analyse the factors that improve short term graft survival[4].

REVIEW OF LITERATURE

The living donor transplantation offers a lot of advantages in terms of graft survival. Ischaemia time and presence of Hypotension / ATN in donor are important determinants of graft survival in cadaver transplants. Living donor transplants are virtually devoid of these adversities and hence we undertake the study to evaluate factors that determine short term graft survival in living donor transplants. The outcome has significantly improved for both cadaver and living donor recipients, but living donor recipients continue to have better long-term patient and graft survival rates (Vs. cadaver donor recipients).

This better outcome was originally attributed to genetic matching; in the past, almost all living donors were relatives. However, many recent studies have noted that living unrelated donor recipients have outcomes similar to those of non-HLA-identical living related donor recipients. Thus, the major advantages of living donor transplants are likely due to the process itself; the ability to evaluate the donor fully, the opportunity to schedule surgery electively when both donor and recipient are in optimal condition, and the minimal ischemic time such that DGF is

relatively rare. In fact, the subset of cadaver donor recipients with excellent immediate post-transplant function have outcomes similar to living donor recipients[5].

Receiving a living (vs. cadaver) donor kidney is a significant advantage. Both short and long-term results are better. A disadvantage of living donation is that donors undergo a major operation that they do not need. Clearly, donors are not better off with one kidney rather than two. However, considerable data support the concept that an individual with one normal kidney can lead a normal life[6]: children born with one normal kidney live a normal life; children or adolescents who have a kidney removed because of a tumor or trauma live a normal life (if the remaining kidney is normal); and donors followed up for 20 to 30 years do not have an increased incidence of kidney disease compared with their brothers and sisters who did not donate[7]. Kidney donors do not have trouble getting life insurance, and insurance rates are not increased after donation. Although donation carries no physical benefit, studies have shown a psychological benefit: an increase in self-esteem.[8] In addition, donor evaluation has revealed previously unrecognised and treatable medical problems.

The biggest challenge in transplantation today is increasing the number of available organs. Although the outcome after living donor transplantation is better than after cadaver donor transplantation, the number of living donor kidney transplants done annually was unchanged for years because of the reluctance of transplant personnel to put a potential donor through a major and unnecessary operation. However, because of the rapidly growing waiting list for cadaver transplants and the increasingly longer wait, most centers are now willing to advocate living donation. In the past 2 years, the number of living donor transplants in the United States has increased. Much of this increase followed the recognition that living unrelated donor recipients had outcomes similar to those of living related non-HLA-identical donor recipients.

Many studies have attempted to identify the risk factors for chronic allograft nephropathy. Most have identified donor related factors such as age and function, and immunologic factors, which are strongly associated with previous acute rejection episodes, as the most important. The effects of potentially nephrotoxic immunosuppressive agents such as calcineurin inhibitors are still widely debated, although there is general

agreement that high levels, even within the first year may pose a threat to long-term function. It is likely that both the etiology of chromic allograft nephropathy and its evolution represent the summation and interaction of multiple variables[9].

For recipients with at least 1-year graft survival, they noted several significant risk factors for worse long-term outcome. Those with pre-transplant cardiac or peripheral vascular disease and those who smoked before the transplant had worse graft survival. Cosio et al[10] recently showed a dramatic decrease in post-transplant patient survival rates in recipients who smoked before the transplant. What can be done to improve the outcome for such recipients? First, those with pre-transplant cardiac or peripheral vascular disease should be aggressively screened to identify any treatable cardiac lesions. If lesions are identified, they should be treated before the transplant. Such an approach has decreased the post-transplant death rate in diabetic transplant recipients. Second, for candidates waiting for a transplant, hyperlipidemia and hypertension should be aggressively managed. Third, if there is a long interval (>1 year) between initial evaluation and the transplant, candidates should

undergo re-evaluation.[11] Finally, all transplant candidates should be strongly encouraged to stop smoking.

The main determinant of 1 year graft survival in the series by Isahl et al, after censoring for death with a functioning graft, was DGF. There were no adverse effects of other donor factors including donor age or ICU management on 1 year graft survival. Importantly, no other factors were significant, including AR, HLA matching, highly sensitized recipients and re-graft. The lack of an effect of AR on 1 year graft survival is surprising but not unexpected. This phenomenon has been mirrored in many recent publications comparing immunosuppressive regiments. It would suggest that AR in the first year is no longer a good endpoint for comparative studies[12].

In a study done by Steven et al, Five year graft survival for 12 months Cr level less than 1 (n=38) was 95% for 1.0 to 1.4 (n=454) 87%; for 1.5 to 1.9 (n=463), 86%; for 2.0 to 2.4 (n=166), 78%; for 2.5 to 2.9 (n=54), 60%; for greater than or equal to 3 (n=45), 41%. A major breakpoint for outcome is 1 year Cr level = 2.0. A power analysis was

performed for the combined endpoint of graft loss and 1 year Cr level greater than 2, reached by 30% of patients[13].

The 12 month serum Cr level was the most powerful predictor of long-term graft failure. Based on this finding, they selected a serum Cr level of 2.0 mg/dL as the point beyond which an excessive decline in 6 and 10 year graft survival was seen. Of recipients with 12 month serum Cr level greater than 2.0 mg/dL 50% lost their graft within 10 years, whereas graft survival was more than 65% at 10 years for those with a 12 month serum Cr level less than 2.0 mg/dL[13].

In a recent publication, Meier-Kriesche and colleagues[14] have reported a strong association between renal function at 1 year and the risk of cardiovascular disease and infectious mortality. According to this publication, a serum creatinine level of 1.9 - 2.1 mg/dl. Conferred a 50% increased risk of cardiovascular death compared with a serum creatiuine level of < 1.3 mg / dl.

The only factors affecting the long-term survival of those grafts that reached 1 year after censoring for death were recipient age and CIT and no apparent effect of AR or HLA matching. Crucially, it was

found in a study by Isahel et al that CIT affects long-term graft survival independently of the phenomenon of DGF. Other studies have found that DGF is one of the most important factors related to graft loss but have not identified CIT as having an impact in the long-term. In contrast, Ojo and colleagues in a study from American registry data found that pro-longed CIT directly and independently of DGF and AR, compromised the long-term graft survival. In this study, it was shown that the effect of CIT on long-term graft survival is linear and hence, there is no threshold below which CIT is acceptable or a threshold beyond which the deleterious affect of CIT accelerates[15].

The serum creatinine at 1 year, rather that the serum creatinine at 3 or 6 months, was found from this analysis to be an excellent predictor of the long-term survival of the graft as was reported by Hariharan[16]. It was also found to be an important factor in a recent report by He and Johnston[17].

Moreso et al have shown that patients receiving a kidney form old donors who suffer from DGF have a very poor long-term graft survival even in rejection free patients. These data indirectly support the suggestion that the deleterious effect of DGF on late graft outcome is

amplified in patients receiving a kidney harvested from an old donor. Consequently it seems reasonable to control those factors associated with DGF such as cold ischaemia time or cyclosporine nephrotoxicity in order to improve long-term results when organs from elderly donors are accepted for transplantation[18].

Female donor gender and higher recipient/donor weight ratio are major predictive factors in the development of DGF following living-related kidney transplantation. Although DGF alone did not affect the outcome, long-term graft survival was significantly reduced when DGF was associated with acute rejection episodes[19].

During the first year post-transplant, the benefits of receiving a living donor kidney (versus a cadaver kidney) mitigate negative cofactor risks of graft failure. Beyond one year, recipients of living donor kidneys are subjected to the same deleterious effects from cofactors and early post-transplant events that impact the long-term graft survival following cadaveric transplantation[20].

In a Study by Nishikawa and Terasaki, using univariate analysis, it was shown that graft survival kidneys from older living

donors was significantly better than that of kidneys from older cadaver donors and was comparable to that of kidneys from younger cadaver donors. Using multivariate analysis, it was shown that the presence of one or more acute rejection episodes significantly shortens both cadaver and living donor long-term graft survival. Although the use of kidneys from cadaver donors >55 years was associated with significantly decreased long-term graft survival, no such association exists for recipients of kidneys from living donors >55 years.

Cold ischaemia has been shown to be especially damaging when the transplantation is performed with a kidney from an elderly donor; DGF appears in over 40% of transplant patients when its cold ischaemia is over 24 h. On the other hand, we do not know the physiopathological mechanism that could explain the relationship between the type of dialysis followed by the uraemia patient and DGF[21]. In our study we could not study the effect of the type of dialysis since all our patients underwent only hemodisalysis.

In the group of patients studied by Ricard et al the presence of DGF in itself only negatively influenced graft survival, after censoring for death, when the kidney came from an elderly donor. However,

considering any kind of donor, they were able to relate DGF to acute rejection and kidney dysfunction, which did demonstrate their negative influence on patient and graft survival. Thus, they detected a greater incidence of acute rejection in the patients that presented with DGF. The mechanism by which DGF and acute rejection are associated has not been completely clarified, but a greater expression of MHC is invoked in the kidneys that suffered from a period of ischaemia, which would more easily provoke the immunological response of the recipient[22].

In a study at Netherlands by Henk Brom et al, several risk factors for DGF were identified, of which a low recipient pre-transplant mean arterial blood pressure, the transplantation of kidneys from female donors to male recipients, and a prolonged cold ischemia time were potentially avoidable. Although DGF is one of the several risk factors of acute rejection and suboptimal function at one year, it is not independently associated with an increased rate of graft loss[23].

In a univariate analysis; DGF was correlated with graft loss within the first year, as were female donor gender, an Aza-based immunosuppressive regimen, CIT of more than 24 hours, and the number and type of rejection episodes. Sharing of HLA class-1 antigens

correlated inversely with graft loss. However, when the data were entered in a multivariate analysis, neither DGF nor cold ischemic time remained a risk factor for graft-loss within the first year. Acute rejection episodes, especially vascular rejection female donor gender and an Aza-based immunosuppressive regimen remained independently associated with graft loss within the first year[23].

Higher grade HLA mismatch has previously been reported to be associated with an increased risk of DGF. Terasaki et al recently showed that graft survival in cadaveric transplants with immediate function is superior to that in zero-HLA mismatched transplants compromised by DGF. The current study by Akinlolu et al showed that zero-mismatched kidneys yielded better graft survival within each category of graft function, but on a comparative basis, the modest benefit of HLA matching is smaller than that early graft function. Thus, it may be better to received a “fresh” kidney than a well-matched one[24].

How can DGF, independent of early acute rejection, negatively effect renal allograft survival? First, severe acute tubular

necrosis may be associated with actual nephron destruction. The development of severe acute tubular necrosis may be further exacerbated by either cytokine-releasing induction therapy and / or cyclosporine therapy if cyclosporine is used before graft function is established. This early pruning of renal nephron mass may predispose to hyperfiltration injury of the remaining nephron mass of the allograft [25], DGF may be associated with a rich tubular-interstitial milieu of proximal proinflammatory agonists, including interferon-γ, interleukin 2, transforming growth factor - β, and interleukin 4 which may stimulate non-antigen-dependent inflammation and scarring.

That DGF is an independent risk factor for long and short-term graft survival is in contract to the results of a single-center study in which DGF was associated with acute rejection episodes, but was not a significant risk factor for diminished 5 year graft survival[26].

The influence of demographic characteristics (age, sex race); transplant variables (cadaver versus living donor, cold ischemia time, HLA mismatching, delayed graft function and transplant year), and post-transplant variables (immunosuppressive agents for the prevention of acute rejection, clinical acute rejection and post-transplant renal function

in the first year) on graft survival were analyzed for 105,742 adult renal transplant between 1988 and 1998.

In conclusion, it was found that one year creatinine and Δ creatinine values predict long-term renal graft survival. Recent improvements in graft half-life are related to conservation of renal function within the first year post-transplantation[27].

In previous studies, discharge creatinine was identified as a strong predictor of transplant survival. The projected median graft half-life for cadaveric transplants with discharge creatinine values of 0.5 to 1.5 mg/dL was 11.5 years. Half-life values for patients with discharge serum creatinine 1.6 to 2.5 ad >2.5 mg/dl were 9.6 and 7.2 years, respectively. But discharge creatinine has limited value as many patients are discharged within a few days after transplant, before they reach nadir creatinine levels. This is true especially for recipients of renal transplants from older donors, those with prolonged cold ischemia time and those who experience DGF. One month creatinine values may be falsely elevated due to higher cyclosporine and tacrolimus levels used to prevent acute rejection. Hence, the study by Hariharan et al, used six month and one year creatinine values to predict long-term graft survival[27].

This study illustrates that event occurring within the first year are of critical importance for long-term graft survival. Thus, the quality of renal function at 1 year should be implemented as a newer endpoint for primary comparative trials.

In a study by Xiang He et al seven independent risk factors for allograft failure were identified; older recipient, male recipient and younger donor above average creatinine chronic allograft nephropathy, diabetic recipient, and neoplasm after transplant.

Among these seven independent risk factors were found to influence graft survival, only two of these could be modified by clinical intervention, elevated serum creatinine at 1 year and the occurrence of chronic allograft nephropathy. To influence these two factors, the optimization of immunosuppressive therapy is essential[29].

CAN (previously often referred to as chronic allograft rejection) is the most important cause of transplant recipients returning to dialysis after a renal transplant. This study confirms that CAN is the main risk factor for graft failure the first year transplantation. Recipients with CAN demonstrated a seven times higher risk of losing their graft

compared with recipients without CAN. Only 70% of patients with CAN still had a surviving graft 7 years post-transplant compared wit h95% of patients free of CAN.

This study confirmed that a higher than average serum creatinine level (i.e., >162 μmol/L) at 1 year post-transplant was linked to poor graft survival. Non immunologic factors of both donor and recipient variables have been widely discussed as risk factors for long-term graft survival. The recipient variables (age, body mass index, sex, history of dialysis) and the donor variables (age, sex cadaveric donor, HLA mismatch, and cold/first warm ischemia time) have all been reported to affect graft survival. In the present study, male recipients had more than twice the risk of demonstrating graft failure than female recipients. Regardless of the different sex combinations of donor and recipient (M/M, M/F, and F/M), female recipients always demonstrated better survival rates than males ($\rho < 0.001$).

Concomitant illnesses have also been shown in previous studies to be risk factors for allograft failure, for example, diabetic recipients have been shown to have a higher risk of graft failure than non-diabetic recipients. This study confirmed that patients with pre-existing

diabetes mellitus demonstrated more than twice the risk of losing their graft than patients without existing disease. However, for those patients who developed diabetes after transplantation, graft survival was not significantly reduced by the onset of the disease. This may, in some part, be because of the quality and frequency of care they receive post-transplantation.

Therefore going though the literature, it is evident that events occurring in the first year of transplant influence graft survival. The S creatinine at 12 month post transplant has been found to be a significant factor determining long term graft survival.

OBJECTIVES OF THE STUDY

To determine the factors that influence graft functioning at 1 year in live related Donor Renal Transplant.

RENAL TRANSPLANTATION IN OUR INSTITUTION

1. Donors:

In our institution, we have so far done 750 Renal Transplantations Only first degree related persons are taken as Donors for renal transplantation. After enquiring the family details, a HLA matching is done. If there is n match, then Approval by Authorisation committee is obtained before transplantation.

At a point of time when kits for HLA matching were not available, the Patients, and Donors were sent to committee only for ascertaining the Relationship.

In our institution the criteria for a Donor is

(i) The person should be more than 20 years and less than 60 years of age.

(ii) The person should either parent sitting or offspring of the patient.

(iii) Spousal Donors are considered provided there is a valid evidence of marriage and when there are no eligible/willing first degree related donors.

Complete evaluations of the Donors are done to R/O any undetected / underlying disease / condition. The Donor should be of perfect health prior to being declared fit for transplantation.

Donor GFR is done using 24 hour urine creatinine estimation. Donors with a Cr cl < 70 ml / minute are rejected.

Donors are tested routinely for Hepatitis Viral serology and HIV ELISA. CMV screening is not done routinely.

Donors are explained in detail about the procedure of transplantations and possible risk even if remote of subsequent Development of Renal Insufficiency.

A written informed consent is obtained from the Donor and the spouse

2. Recipient:

Recipient can be of any age. The recipient is initially evaluated fully. The cause of CKD is ascertained in as many cases as possible. If the patient has a kidney size amenable to Biopsy a tissue diagnosis is obtained.

All recipients undergo viral serology testing. They undergo complete cardiac / gastroenterology / ENT / Dental and dermatological evaluation. All patients are given 3 doses of Double dose HBV vaccination.

Any forms of sepsis are treated before the transplantation procedure.

A voiding cystourethrogram is done for all patients to exclude lower urine tract anomalies.

All patients are maintained only on Haemodialysis prior to transplantation.

All patients are given triple immunosuppression with cyclosporine, azathioprine and prednisolone.

MATERIALS AND METHODS

This is a retrospective study.

The patients who underwent Live Donor Renal Transplantation between March 2005 to February 2007 were taken for study.

Demographic data such as Name, Age gender of both Donor and Recipient were recorded.

The other variables taken were for the Recipient.

Native Kidney Disease

Blood group

HLA typing

Cardiac / Respiratory status.

Presence/Absence of

Diabetes Mellitus

For the Donor : Relationship

GFR

HLA typing

Blood group

Common Parameters : Gross matching.

Intra Operative and Post operative data :

Intra operative Hypotension

Cold Ischaemia Time

Warm Ischaemia Time

I day urine output

I day S-creatinine

Time to Normal creatinine

Discharge creatinine

Creatinine during 3, 6 & 12 months.

Events during I year

AR / GDF /Infections

Biopsy of Allograft if done

All the data were fed into a master chart and statistical analysis were done.

EXCLUSION CRITERIA

All the patients who did not survive beyond the first year of transplant were not included in the study.

The patient who underwent graft nephrectomy for graft artery thrombosis was not included.

RESULTS

Demographic Data:

Totally 89 Renal transplantations all live related Donor transplantations took place from March 2005 to February 2007.

Patients among them died within the first year and were not included. One person underwent graft nephrectomy due to graft artery thrombosis. After excluding 10 such patients, 79 patients were included in the study.

The Recipients age ranged from 13 years to 51 years. The mean age of the Recipients was 28.58 ± 8.59 years.

The Donors' age ranged from 24 years to 58 years. The mean age was 44.5 ± 7.81 years.

Among the recipients, there were 68 males. There were only 14 males among the Donors. Recipients were predominantly male and Donors were mostly Females.

Age Distribution: Recipient

Mean	28.58 Yrs.
S.D.	8.59 Yrs.
Min	13 Yrs
Max	51 Yrs.

Donor Age Distribution:

Mean	44.5 Yrs.
S.D.	7.81 Yrs.
Min	24 Yrs
Max	58 Yrs.

Recipient – Gender Distribution:

Particular	No.	%
Male	68	86.1
Female	11	13.9
Total	79	100

Donor – Gender Distribution:

Particular	No.	%
Male	14	17.7
Female	65	82.3
Total	79	100

The native kidney diseases in the transplanted patients were as follows

Native Kidney Disease:

Particular	No.	%
Diab Neph.	1	1.3
CGN	20	25.2
RPGN	1	1.3
FSGS	2	2.5
DPGN	1	1.3
PUV	2	2.5
CIN	1	1.3
IGAN	6	7.5
Obs. Neph	2	2.5
N/K	43	54.4

The nature of HLA matching were as follows:

HLA Matching:

Type	Frequency	%
Nil	0	0
Haplo	60	75.8
Full House	3	3.8
ND	16	20.4

HLA matching could not be done for 16 patients/donors since the kit was not available at that time.

OUT COMES

Events during the first year were as follows:

	No	%
None	43	54.4
AR	13	16.3
GDF-CNI	2	2.5
GDF-CMV	4	5.2
GDF-CAN	4	5.2
GDF-UTI	3	3.8
GDF - uncl.	10	12.6

Graft Function at 1 year:

S. Creat	Frequency	%
< 106 μmol/L	21	26.6
107 to 176	38	58.1
> 176	20	25.3

One year graft function with respect to the following variables were analysed:

Graft Function with respect to:

Donor Age:

Age in years	S Creat ≤ 176	S. Creat > 176	Total
20 - 30	39	13	52
31 - 40	12	5	17
41 - 50	7	2	9
51 - 60	1	--	1
Total	59	20	79

P value < 0.05 significant

Donor GFR:

GFR ml/min	S cr ≤ 176	S cr > 176	Total
70 – 80	7	8	15
81 – 90	20	3	23
91 – 100	16	3	19
> 100	16	6	22
Total	59	20	79

Donor GFR was found to be a significant variable.

Cold Ischaemia Time:

CIT (min)	S cr ≤ 176	S cr > 176	Total
< 30	3	--	3
31 - 40	29	10	39
41 - 50	21	7	28
> 50	6	3	9
Total	59	20	79

P value not significant

Inta Operative Hypotension:

I O Hypotension	S cr $\leq$ 176	S cr > 176	Total
Nil	52	14	66
Present	7	6	13
Total	59	20	79

$X^2 = 3.84$

P = 0.05 - significant

I day Urine Output:

I day Output (ml)	S cr ≤ 176	S.cr > 176	Total
< 4000	3	3	6
4000 - 6000	7	2	9
6000 - 8000	10	2	12
8000 - 10000	17	2	19
> 10000	22	11	33
Total	59	20	79

Time to (N) Creatinine:

Duration (Days)	S cr ≤ 176	S. cr > 176	Total
< 3 days	25	6	31
3 - 7 days	19	7	26
> 7 days	15	7	22
Total	59	20	79

P value not significant.

Discharge Creatinine:

Disch. S.Creat (μmol/L)	1 year S Creat ≤ 176	1 year S. Creat > 176	Total
< 88	21	4	25
89 - 106	29	4	33
> 106	9	12	21
Total	59	20	79

$X^2 = 15.44$

P = 0.001 – significant

Analysis of variables influencing the S.Creat at 1 year:

Mean creatinine with respect to events in the first year:

Events in I year	No.	Mean S Creat	SD
None	43	115.20	22.385
AR	13	273.80	82.561
GDF-CNI	2	237.00	4.243
GDF-CMV	4	180.75	48.083
GDF-CAN	4	334.00	137.179
GDF-UTI	3	191.00	74.246
GDF - uncl.	10	213.60	104.858

Oneway Anova – Analysis of variance

F = 8.74

P = 0.001 - significant

Factors influencing graft function at 1 year

Factors	S cr ≤ 176		S. cr > 176		P Value
	Mean	SD	Mean	SD	
Age of rec.	28.27	9.1	29.50	6.8	0.58
Donor Age	43.12	7.4	48.75	7.7	0.001
CIT	43.66	8.6	45.25	6.4	0.47
I day output	92.08	3364	9736	4487	0.58
Time to N creat	5.58	4.2	5.84	3.9	0.88
Dis Creatin	100.44	20.6	131.40	66.8	0.002
Donor GFR	92.90	12.1	90.20	14.1	0.47

Donor Age and Discharge creatinine influenced graft fn at 1 year significantly.

Multivariate Analysis by Cox Regression Model.

Factors	B	SE	Wald	Df	Sig	Exp (B)
Donor Age	- .048	.016	8.506	1	.004	.953
Donor GFR	.005	.011	.191	1	.662	1.005
Hypotension	.502	.409	1.504	1	.220	1.652
Dis. Creat	- .047	.183	.067	1	.796	.954

By multivariate analysis Donor age was the most significant factor influencing graft function at 1 year.

DISCUSSION

Among the 89 Renal transplantations done during the study period, 79 were taken for the study. Ten patients were not included one person had a steroid resistant rejection, never regained graft function and died due to Sepsis; one person had respiratory failure secondary to status epilepticus. One patient had a graft artery rupture; five patients died due to fulminant sepsis; one patient with ADPKD had graft artery stenosis with partial recanalisation and subsequently succumbed to sepsis. The tenth patient had graft nephrectomy for graft artery thrombosis and returned to dialysis.

The patients old records were analysed to study the nature of the native kidney disease. The native kidney disease could not be found out in 43 out of 79 patients included in the study.

There were two patients with type 2 diabetes Mellitus. One of them had Diabetic Nephropathy leading to chrome Kidney Disease. The other person presented with no proteinuria and contracted kidneys and

therefore a diagnosis non diabetic kidney disease was diagnosed in that patient.

Two patients had Posterior Urethral values which was diagnosed in childhood and treated but patients later progressed to chronic kidney Disease. They had fulgration of the remaining value leaflets prior to surgery with no Bladder outlet obstruction.

One patient had chronic Interstitial Nephritis. One more patients had a spinal trauma followed by neurogenic bladder. He required an Ileal conduit which was done 3 months prior to the transplantation.

All the other patients had chronic glomerulo nephritis as judged by the presence of proteinuria, borderline size kidneys, Oedema and severe hypotension. We were above to do Biopsy in ten o these patients.

One patient had presented as Rapidly progressive Glomerulo Nephritis with renal Biopsy showing crescentic GN. This patient had cytotoxic therapy but was dialysis dependent and ultimately had End stage renal disease.

One patient presented with Nephritic Nephrotic Syndrome and had Diffuse Proliferative GN, with crescents on Renal Biopsy. There was a rapid progression to ESRD inspite of cytotoxic therapy.

Two patients had focal segmental glomeruls Sclerosis on initial Biopsy. They did not respond to steroid therapy and progressed to ESRD one over a period of 5 years and the other over 6 years.

Six patients had chromic Ig A Nephropathy by renal Biopsy.

The rest were diagnosed as chromic GN by clinical, biochemical and Radiological parameters as outlined above.

All the Donors were first degree relatives except one of them – who was a spousal Donor Tissue typing was done whenever it was available in the Hospital. It could not be done for 16 patients and Donors. Authorisation Committee approval was obtained for these patients.

The other 63 patient / Donors underwent tissue typing. None of them had nil match 60 patients had Haplomatched Donors and 3 patients had full house match.

On analysing the Events which took pace in the first year, 43 patients had uneventful period. There were 13 patients who had biopsy persons Acute Rejection. They were given Pulse-Methyl Prednisolone.

Four patients ha CMV infection as proved by the presence of B 65 antigen and Graft Dysfunction. They were treated with Ganciclovir.

Four patients had Graft dysfunction with evidence of chromic Allograft Nephropathy on Renal Biopsy.

There patients had Urinary tract infection and graft dysfunction due to graft pyelonephrits.

Two patients had high cyclosporine levels with evidence of CNI toxicity on Renal Biopsy.

Ten patients had Graft dysfunction but were not willing for graft biopsy due to logistic reasons. They were given pulse Methyl preduisolone with a clinical diagnosis of Acute Rejection Graft function at the end of the first year was divided into 2 groups. Those patients who had a S-Creatinine of > 176 μmol/L (2mg/dl) and those who had a S.creatinine of ≤ 176 μmol/L.

59 patients our of 79 (84.7%) had S.creatinine levels less than 176 μmol/L. Out of them 21 had S.Creatinine levels less than 106 μmol/L. (26.6%)

On analysing the variables that affect graft function at 1 year the following observations were made:

1. In the younger Donor age group, a significant number of patients had a S.creatinine of less than 176 μmol/L. In a study by Moreso et al it was found that patients receiving kidney from an older donor and suffer from DGF have a very for long term graft survival .

2. It was also observed that a good proportion of patients whose donors had higher GFR and a S.creatinine level less than 176 μmol/L. The results were statistically significant by the χ^2 and student t test.

3. Merely 50% of those who had Intra operative hypotension, had S.creatinine levels >176 μmol/L versus only 28% of those who did not have hypotension had high S.creat. These values were not

statistically significant due to the small number of patients who had intra operative hypotension.

4. The first day urine output and the time taken to reach normal creatinine di not affect the 1 year graft function significantly.

5. Discharge creatinine was found to have a very good association with creatinine at 1 year. Only 15% of those who were discharged with normal creatinine had S.creatinine >176 μmol/L at 1 year, compared to 58% of those who were discharged with more than normal S.creatinine values. This was statistically significant.

6. The Average S.creatinine at 1 year was significantly different between those patients who had an uneventful period and those who had Graft dysfunction during that period (first year of transplantation). Persons who were diagnosed to have chromic Allograft Nephropathy by Biopsy and those persons who had Acute Rejection episodes had worst graft functions. Patients with CMV Infection or UTI had better graft function. These differences were statistically significant.

On further analysis by one way ANOVA method, it was found that the mean GFR of Donors I patients who had a S.creatinine at 1 year of <176 μmol/L was 92.90 ml/min ± 12.1 as against 90.20 ml/min ± 14%.

This was not found to be statistically significant.

The cold ischaemic time in the two groups were also not different to cause statistical significance.

In these two instances it is important to identify that in this study, the Donors are live related where, the GFR is found to be within a normal range (since donors having poor GFR are rejected) and also the cold Ischaemia time is subsequently short when compared to cadaver Donor kidney transplants.

The age of the recipient, first day output and time to normal creatinine were not found to be affecting graft function significantly.

The mean age of the donors whose recepients' creatinine at 1year ≤ 176 μmol/Lwas 43.12 ± 7.4 years; versus 48.75 ± 7.7 years in those who had 1 year S.creatinine > 176 μmol/L. This difference was statistically significant.

The mean discharge creatinine was 100.44 ± 20.6 μmol/L in patients having 1 year S.creatinine ≤ 176 μmol/L. whereas it was 131.40 ± 66.8 μmol/L in those who had 1 year S.creatinine of > 176 μmol/L. This difference was statistically significant.

On Multivariate Analysis using Cox regression method, only Donor Age was found to be the significant variable affecting graft function at 1 year, the P value being 0.004.

It is probable that younger Donor had better GFR and the mean discharge creatinine was lower in those patients who received kidney from younger donors.

CONCLUSIONS

1. Donor age influenced graft function at 1year significantly.

2. Discharge creatinine influenced graft function significantly.

3. Donor GFR was significantly associated with graft function at 1 year.

4. Even though Intra operative hypotension had an influence on graft function at 1 year, it was not statistically significant.

5. Events such as Acute Rejections, and Graft Dysfunction during the first year significantly influenced graft function at 1 year.

6. Recipient Age, First day urine output and time taken to reach normal creatinine post operatively were not significantly associated with graft function at 1 year.

LIMITATIONS

This study had only analysed living Donor transplantations. No cadaver transplants wee analysed.

All patients were given the same Immuno-suppression, making comparison between various Immuno-suppression drugs impossible.

Graft biopsies could not be done and CNI levels could not be tested for all patients with graft dysfunction due to logistic reasons.

STATISTICAL METHODS

The following Statistical methods were used.

- Student t' test
- Chi square test
- Analysis of variance by one way ANOVA method
- Cox regression model for Multivariate analysis.
- P value of < 0.05 is considered significant.

DEFINITIONS

- Normal S.creatinine means a value of S.creatinine less than 106 μmol/L.
- Graft Dysfunction means an acute rise in serum creatinine with or without decrease in urine output.
- Acute Rejection has been defined when there is graft dysfunction with graft biopsy findings at Acute Rejection.

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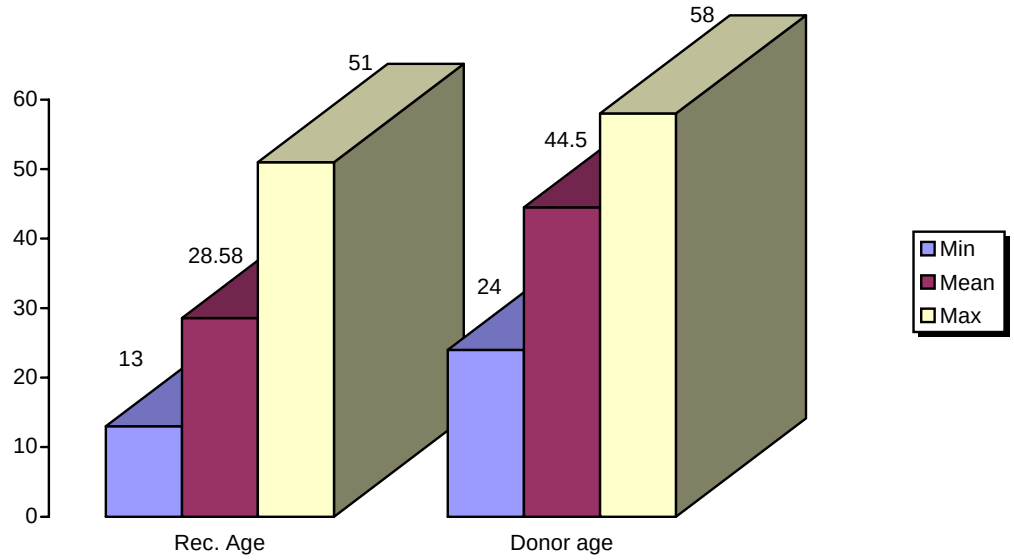
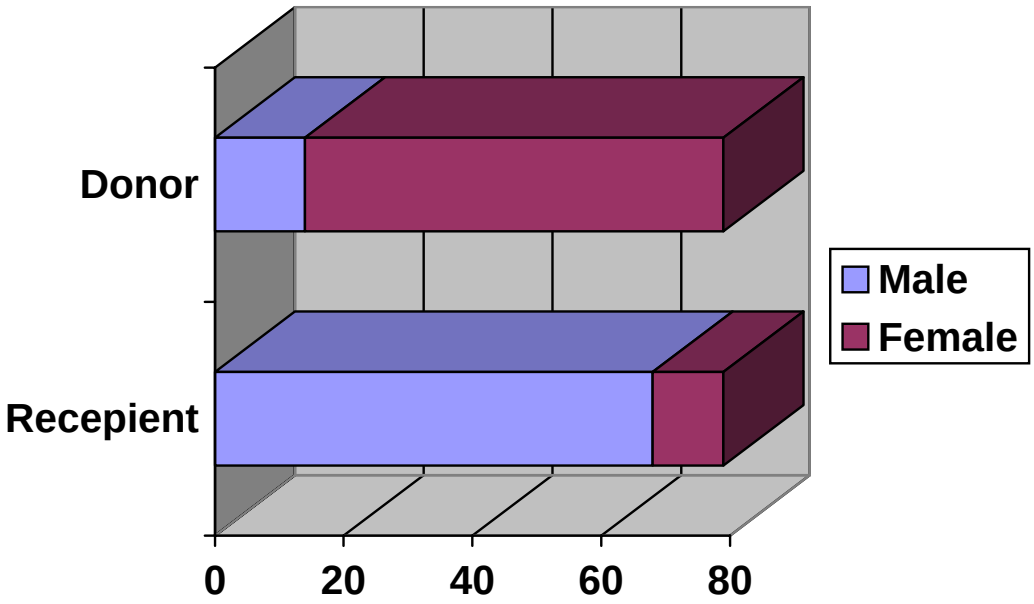
ANNEXURE - I

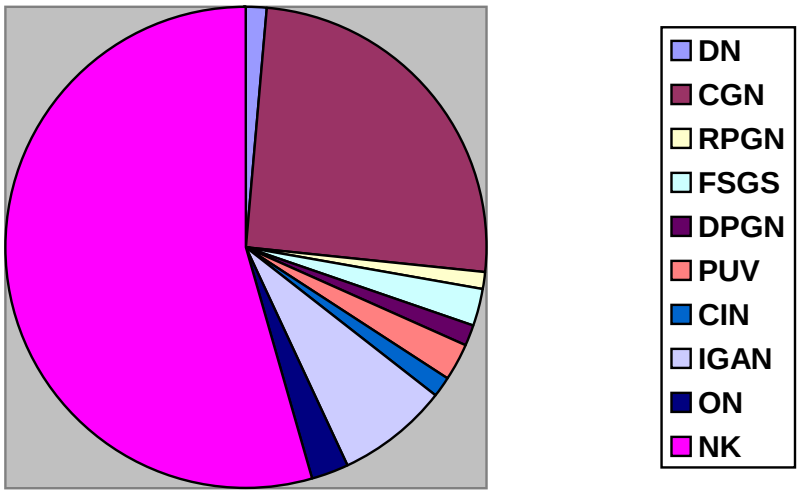
Age distribution of Donor and recipient:



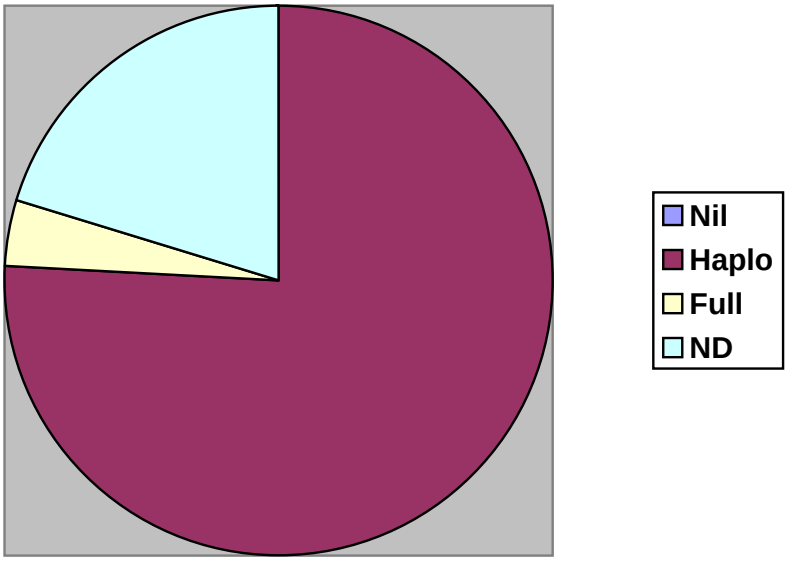
Gender distribution among donor and recipients:



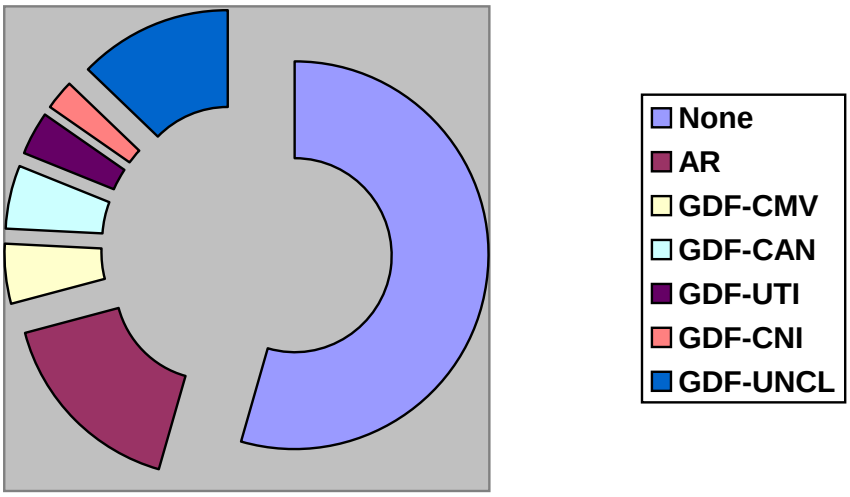
Distribution of Native Kidney disease:



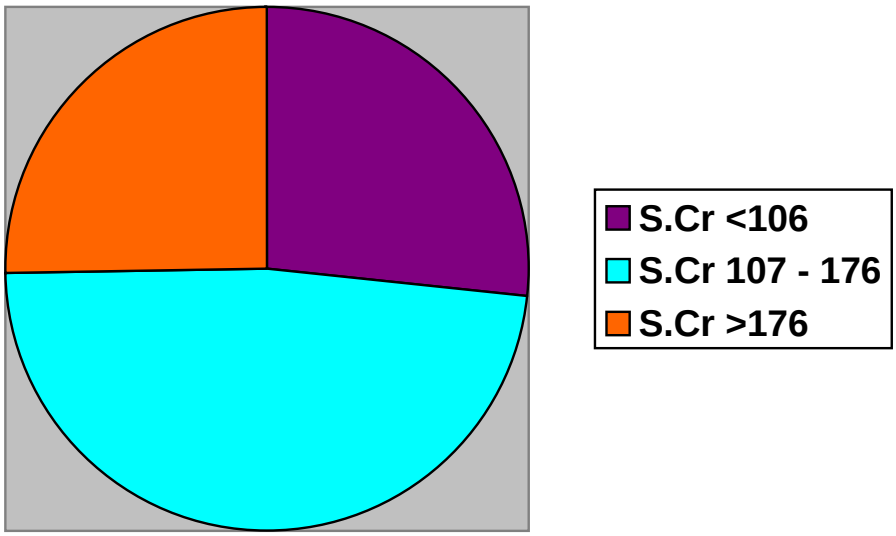
HLA matching of Donor and Recepient.



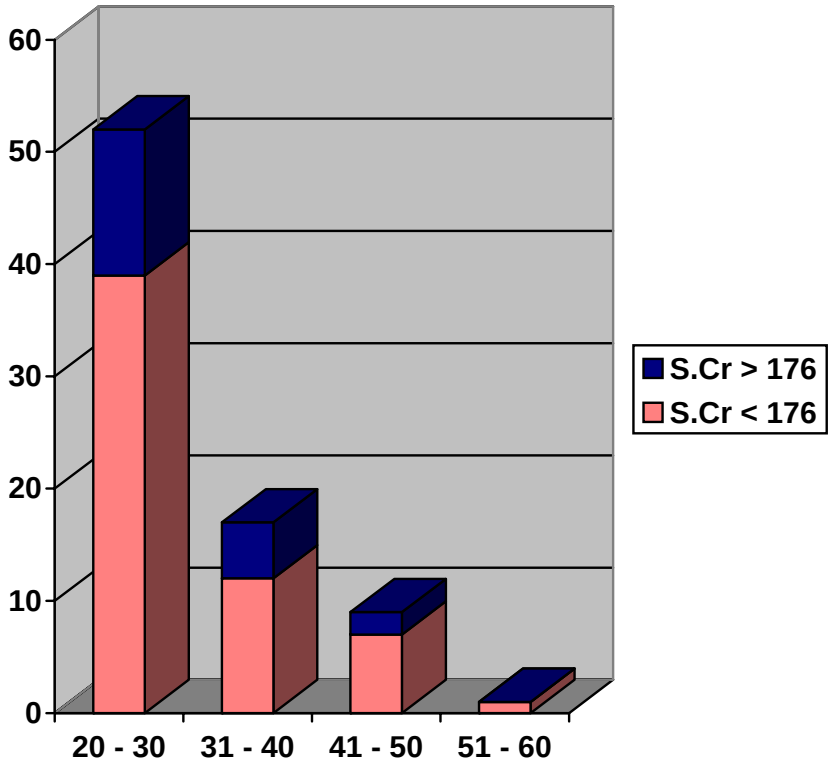
Events distribution in First year:



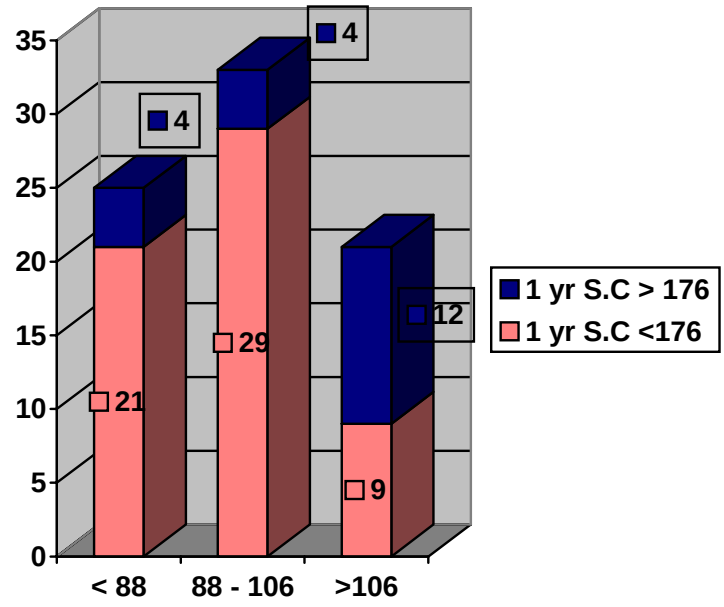
Graft function at 1 year:



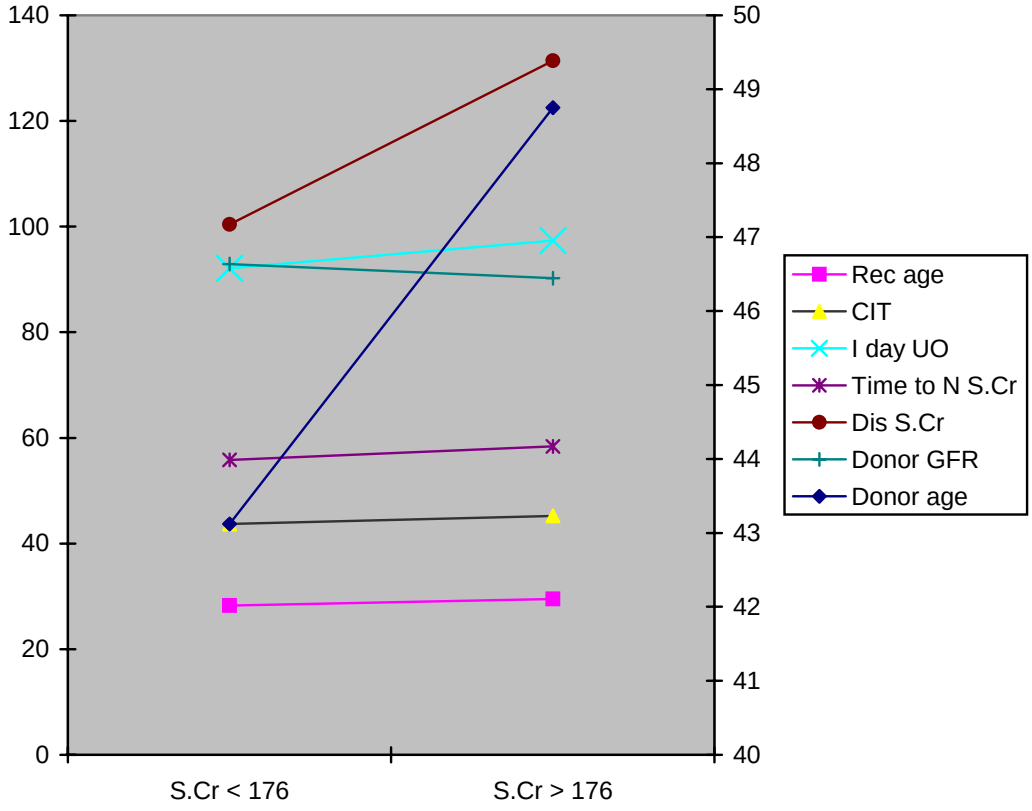
Graft function with respect to Donor age groups:



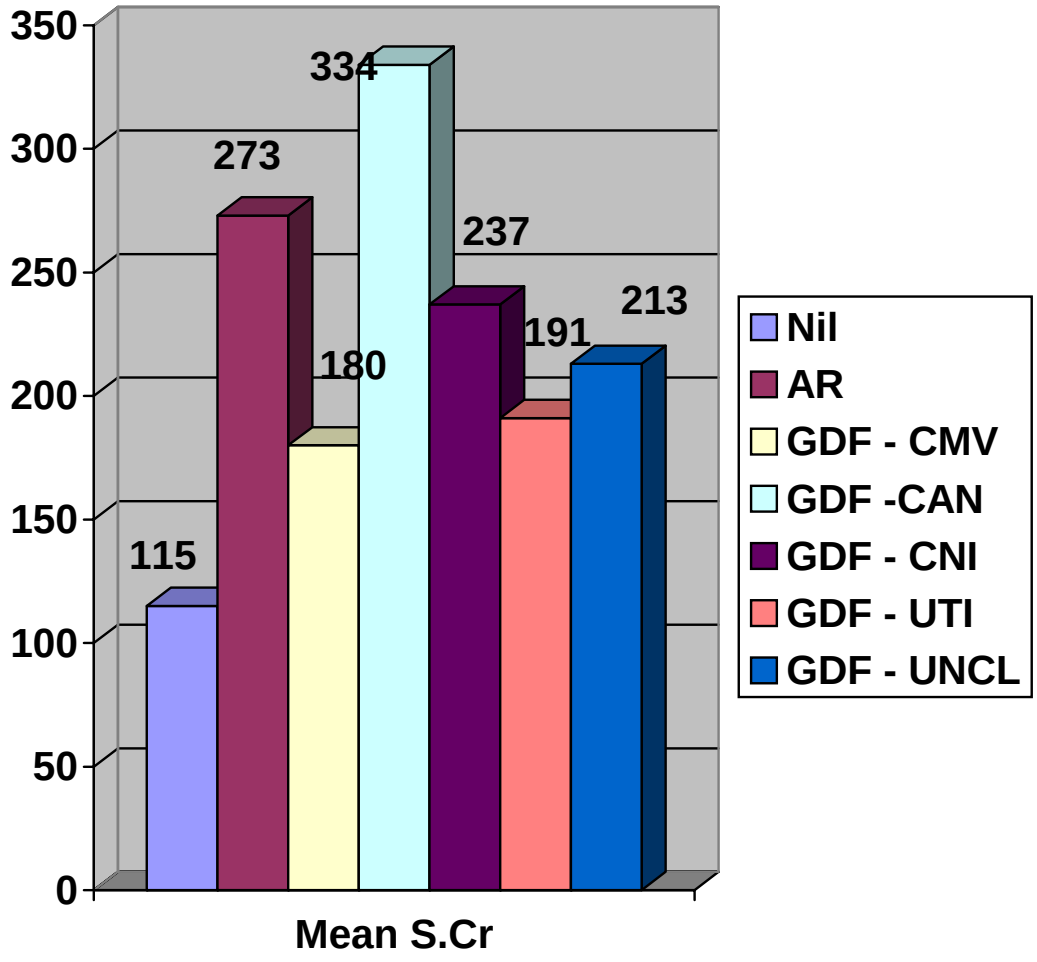
Graft function at one year with respect to discharge creatinine:



Univariate analysis of factors influencing graft function at 1 year:



Mean Se. Creatinine at the end of one year with respect to events occurring in the first year:



Sl. No:	Name	Age	Gender	NKD	DM	Bl.Gr	D age	Gender	Rel	GFR	HLA match	CM	donor BG	Intra-op hypo	CIT	WIT	1st day UO	1st day creat	Time to N. creat. in days	Dis creat	3rd mo creat	6th mo creat	12th mo creat	SGF/ DGF	CNI tox	Surg prob	GDF/AR/AR T	Infections	Biopsy
1	prabu	20	M	CGN	0	B+ve	55	M	F	96	3\6	5%	O+ve	nil	45	3	8800	396	21	114	149	142	140	SGF	Nil	nil	Nil	HCV +ve	ND
2	kumar	40	M	IG AN	0	AB+ve	58	F	M	79	3\6	5%	B+ve	nil	40	2	16500	246	10	148	156	228	311	SGF	Nil	nil	AR Pulsed	UTI I MO	AR
3	veeramani	30	M	NIL	0	O+ve	35	F	S	80	3\6	5%	O+ve	nil	40	3	12300	184	8	114	130	148	196	N	Nil	nil	GDF	nil	ND
4	mahesh	27	M	IGAN	0	B+ve	54	F	M	84	3\6	5%	B+ve	nil	45	3	5980	237	14	131	144	160	200	SGF	Nil	nil	GDF -CMV	CMV +ve	Normal
5	divya	19	F	NIL	0	O+ve	43	F	M	104	3\6	10%	O+ve	PRESENT	40	1	8300	175	3	88	90	88	88	N	Nil	nil	Nil	nil	ND
6	venkatesh	31	M	FSGS	0	O+ve	50	F	M	84	4\6	5%	O+ve	nil	80	5	14450	102	2	88	95	105	123	N	Pos	nil	Nil	can	ND
7	murugan	42	M	CGN	0	A+ve	51	F	S	86	3\6	5%	A+ve	PRESENT	50	3	5000	254	10	88	88	88	130	DGF	Pos	nil	GDF -CMV	CMV +ve	ND
8	velusamy	32	M	NIL	0	A1+ve	50	F	M	98	3\6	10%	A+ve	nil	40	3	9250	140	7	95	88	114	166	N	Nil	nil	Nil	Viral wart	ND
9	pandiaraj	17	M	NK	0	O+ve	45	F	M	88	3\6	10%	O+ve	nil	40	1	11108	184	2	88	88	140	160	N	Nil	nil	Nil	nil	ND
10	murugan	27	M	NIK	0	B+ve	50	F	M	98	3\6	5%	B-Ve	nil	50	1	15700	108	3	88	88	80	80	N	Nil	nil	Nil	nil	ND
11	padmanaban	20	M	NIL	0	O+ve	50	F	M	90	3\6	5%	O+ve	nil	50	1	10750	108	4	88	88	88	115	N	Nil	nil	Nil	nil	ND
12	rajan	32	M	NIL	0	O+ve	26	F	W	88	ND	5%	O+ve	PRESENT	40	4	12200	96	3	88	88	88	80	N	Nil	nil	Nil	Otitis	ND
13	dandayuthabani	43	M	DN	Pr	O-ve	55	F	S	80	ND	10%	O+ve	nil	30	3	3070	254	10	105	96	104	103	SGF	Pos	nil	Nil	Pulmucor	ND
14	bala murugan	20	M	NIL	0	B+ve	35	F	M	105	3\6	5%	B+ve	nil	30	3	11000	149	11	90	88	80	156	N	Pos	nil	AR Pulsed	Cellulitis	AR
15	subburathinam	31	M	CGN	0	B+ve	24	F	S	110	ND	5%	B+ve	nil	40	3	11650	175	2	88	96	88	123	N	Nil	I&D	Nil	Folliculitis	ND
16	balaji	19	M	RPGN	0	B+ve	40	M	F	82	3\6	10%	B+ve	nil	50	3	5300	156	2	88	88	90	175	N	Pos	Nil	GDF -CMV	nil	ND
17	manoharan	51	M	NK	Pr	A+ve	42	M	B	78	NIL	5%	A+ve	PRESENT	35	2	7750	123	8	96	90	96	104	N	Nil	nil	Nil	nil	ND
18	ezrilarasi	13	F	NIK	0	A+ve	35	F	M	102	3\6	5%	A+ve	PRESENT	40	3	2000	262	10	105	96	88	132	SGF	Nil	LYM	Nil	nil	ND
19	gokulnath	20	M	NIK	0	B+ve	45	M	F	110	Nil	10%	B+ve	nil	50	5	1400	490	13	260	306	480	623	DGF	Nil	GAT	CAN	nil	CAN
20	vivekanandan	32	M	CGN	0	A2+ve	55	F	M	105	3\6	10%	O+ve	nil	40	3	7550	210	4	88	88	254	229	N	Pos	nil	AR Pulsed	nil	AR
21	b.v.baskar	33	M	CGN	0	AB-ve	38	F	S	45	Nil	5%	O+ve	nil	40	3	12000	130	10	88	87	96	105	N	Nil	nil	Nil	UTI	N
22	sasikumar	24	M	NIK	0	AB+ve	48	F	M	98	Full	10%	AB+ve	nil	40	3	8000	148	3	88	96	271	134	N	Pos	nil	AR Pulsed	PT, HCV	AR
23	sukumar	24	M	NIK	0	B+ve	30	F	S	110	3\6	10%	B+ve	PRESENT	40	2	9000	96	1	88	131	219	440	N	Nil	nil	GDF -CAN	UTI, cellulitis	CAN
24	sakthivel	35	M	NIK	0	A+ve	56	F	M	106	3\6	10%	A+ve	nil	40	2	16000	100	2	96	150	302	286	N	Nil	GRA	AR Pulsed	PT,CMV	AR
25	lakshmi	27	F	CGN	0	A+ve	50	F	M	98	3\6	10%	A+ve	PRESENT	50	2	14000	118	4	88	120	228	340	N	Nil	nil	GDF -CAN	nil	Early CAN
26	shiek fareed	26	M	NIK	0	B+ve	48	F	M	105	3\6	5%	B+ve	PRESENT	50	3	6300	190	3	99	86	148	131	N	Nil	nil	AR Pulsed	CMV, TB	ACR
27	govindraj	37	M	FSGS	0	B+ve	56	F	M	74	3\6	0%	B+ve	nil	50	4	8750	280	18	104	154	148	140	SGF	POS	nil	Nil	HBV, LRI	Nil
28	kumari	36	F	NIK	0	O+ve	48	F	S	76	3\6	5%	O+ve	nil	50	2	9100	237	12	123	123	208	190	SGF	Nil	nil	Nil	HIV, UTI	ND
29	kulandaivelu	38	M	NIK	0	O+ve	36	M	B	102	3\6	10%	O+ve	nil	40	3	11450	105	2	87	88	140	162	N	Pos	nil	GDF	HCV +ve	ND
30	m.s.ravi	49	M	NIK	0	O+ve	40	M	B	103	ND	5%	O+ve	nil	40	2	10000	148	1	88	88	88	88	N	Nil	nil	Nil	nil	Normal
31	manikandan	19	M	NIK	0	O+ve	45	F	M	84	3\6	10%	O+ve	nil	45	3	6200	131	3	96	123	134	136	N	Nil	nil	Nil	nil	ND
32	selvaraj	32	M	NIK	0	O+ve	55	F	M	82	3\6	5%	O+ve	nil	40	2	7550	96	8	114	148	166	168	N	Nil	nil	AR Pulsed	UTI	ACR
33	sharmila	25	F	NIK	0	B+ve	45	F	M	98	3\6	5%	B+ve	nil	45	3	13350	148	2	88	156	262	146	N	Nil	nil	GDF	nil	ND
34	suresh	22	M	NIK	0	AB+ve	55	F	M	72	ND	5%	A+ve	PRESENT	40	3	13400	131	5	88	140	141	261	N	Nil	nil	GDF-CMV	UTI, CMV	ND
35	chinna	26	M	NIK	0	AB+ve	47	F	M	78	4\6	10%	B+ve	nil	45	3	5200	289	8	114	148	209	140	SGF	Nil	nil	AR Pulsed	LRI	ND
36	udayakumar	42	M	DPGN	0	B+ve	56	M	F	74	ND	5%	B+ve	PRESENT	50	3	12400	131	3	96	343	298	314	N	Pos	nil	GDF	nil	ND
37	veerapandian	20	M	PUV	0	B+ve	40	F	M	82	ND	5%	B+ve	nil	45	3	7200	184	10	148	230	164	148	SGF	Nil	nil	Nil	nil	ND
38	veeramani	30	M	CKD	0	O+ve	35	F	S	80	3\6	5%	O+ve	nil	40	5	12300	184	6	114	166	168	238	N	Nil	nil	AR Pulsed	ADD	ACR
39	mahesh	22	M	CGN	0	B+ve	44	F	M	84	4\6	5%	B+ve	nil	45	3	5900	237	16	131	148	154	132	SGF	Pos	nil	GDF -CMV	CMV	ND
40	senthil kumar	24	M	GN	0	B+ve	45	F	M	77	ND	5%	O+ve	nil	40	3	13700	148	6	85	131	178	134	N	Pos	nil	GDF	LRI	ND
41	siva kumar	25	M	NIL	0	AB+ve	45	F	M	94	3\6	10%		PRESENT	35	3	8060	210	5	105	146	140	124	N	Pos	nil	GDF	nil	ND
42	vijayalakshmi	20	F	CGN	0	O+ve	55	M	F	70	ND	5%	O+ve	nil	60	3	5750	140	4	105	85	120	240	N	Pos	nil	GDF- CNI	nil	CNI tox
43	ganesh kumar	41	M	NIK	0	B+ve	47	M	B	74	6\6	5%	B+ve	nil	40	3	10500	140	2	96	87	105	110	N	Nil	nil	Nil	CMV	ND
44	rameshkumar	37	M	CGN	0	O+ve	42	F	S	104	4\4	5%	O+ve	nil	35	3	10000	105	3	88	96	105	98	N	Nil	nil	Nil	Tonsillitis	ND
45	shah nawaz	20	M	NIK	0	O+ve	45	F	M	89	ND	5%	O+ve	nil	35	3	11000	356	10	184	140	108	110	SGF	Nil	nil	AR Pulsed	UTI	ND
46	rajagopal	25	M	IGAN	0	O+ve	46	F	M	89	5\6	5%	O+ve	nil	40	3	12300	193	4	123	136	268	234	N	Pos	nil	GDF	UTI	CNI tox
47	rajesh	22	M	CGN	0	B+ve	38	F	M	89	5\6	5%	O+ve	nil	40	3	8400	123	3	96	88	100	86	N	Nil	nil	Nil	nil	ND
48	arul	30	M	)bs. Nep	0	B+ve	47	F	M	82	3\6	10%	O+ve	nil	40	3	9850	140	2	105	115	138	152	N	Nil	nil	Nil	RecUTI	ND
49	kumar	40	M	NIK	0	AB+ve	35	F	S	90	3\6	10%	AB+ve	nil	40	3	11650	88	1	86	85	88	84	N	Nil	nil	Nil	CMV	ND
50	ayyappan	44	M	IGAN	0	B+ve	41	M	B	112	3\6	5%	O+ve	nil	50	3	6600	200	9	114	115	112	103	SGF	Nil	nil	Nil	nil	ND

Sl. No:	Name	Age	Gender	NKD	DM	Bl.Gr	D age	Gender	Rel	GFR	HLA match	CM	donor BG	Intra-op hypo	CIT	WIT	1st day UO	1st day creat	Time to N. creat. in days	Dis creat	3rd mo creat	6th mo creat	12th mo creat	SGF/ DGF	CNI tox	Surg prob	GDF/AR/AR T	Infections	Biopsy
51	kamaldas	22	M	NIK	0	A+ve	26	M	B	91	3\6	5%	A+ve	nil	30	3	15850	96	2	86	105	166	120	N	Nil	nil	Nil	PT	ND
52	kamala	27	F	NIK	0	A1-ve	46	F	M	91	3\6	10%	A1-ve	nil	50	3	10600	184	5	105	97	114	115	N	Nil	nil	Nil	nil	ND
53	jagadesan	20	M	CGN	0	A-ve	50	F	M	104	4\6	5%	O+ve	nil	43	2	14700	123	4	86	96	150	151	N	Pos	nil	GDF	nil	Normal
54	saravanan	33	M	CGN	0	O+ve	27	F	S	98	3\6	15%	O+ve	nil	55	2	6100	184	4	105	96	121	130	N	Nil	nil	Nil	nil	ND
55	siva kumar	28	M	NIK	0	B+ve	50	F	M	106	3\6	10%	B+ve	nil	55	3	11600	105	3	123	281	260	280	N	Nil	nil	GDF -UTI	UTI	ND
56	komala	28	F	NIK	0	O+ve	55	F	M	88	4\6	5%	O+ve	nil	55	2	6200	123	5	96	85	126	175	N	Nil	nil	GDF -UTI	UTI	ND
57	saravanan	28	M	NIK	0	O+ve	46	F	M	106	4\6	5%	O-ve	nil	45	3	19100	153	3	96	113	143	130	N	Nil	nil	Nil	HCV+ve	ND
58	arunkumar	19	M	Obs. Nep	0	B+ve	36	F	M	112	3\6	5%	O+ve	nil	65	3	2500	325	14	146	198	122	119	SGF ATI	Nil	nil	GDF UTI	UTI	ND
59	vel murugan	13	M	PUV	0	A+ve	45	F	M	96	3\6	5%	A+ve	nil	45	2	5150	156	5	88	103	122	124	N	Nil	nil	Nil	UTI	ND
60	arun	32	M	NIK	0	B+ve	54	F	M	74	3\6	5%	B+ve	PRESENT	45	3	850	609	N A	367	265	283	234	SGF	Nil	nil	GDF -CAN	nil	Early CAN
61	xavier	25	M	CGN	0	A1-ve	45	F	M	93	3\6	15%	A1-ve	nil	47	3	11900	155	6	89	103	99	101	N	Nil	nil	Nil	nil	ND
62	nagaiah	27	M	CKD	0	B+ve	32	M	B	98	3\6	5%	O+ve	nil	42	3	8700	108	2	94	100	104	98	N	Nil	nil	Nil	nil	ND
63	ponnusamy	40	M	NIK	0	A+ve	36	F	S	88	3\6	5%	O+ve	nil	60	3	10700	150	7	93	110	144	124	N	Nil	nil	Nil	HBV	ND
64	shanmugan	46	M	NIK	0	A1+ve	40	F	S	90	3\6	5%	O+ve	nil	60	3	4800	144	5	98	134	248	144	N	Nil	nil	AR Pulsed	nil	ND
65	Buvaneswaran	23	M	NK	0	O+	48	F	M	98	3\6	5%	O+ve	Nil	45	3	8700	235	4	106	98	90	96	N	Nil	Nil	Nil	Nil	ND
66	Jayanthi	30	F	NK	0	B+	38	M	B	116	3\6	5%	O+ve	Nil	40	3	9600	214	3	98	96	90	88	N	Nil	Nil	Nil	Nil	ND
67	Rameshkumar	24	M	CGN	0	B+	49	F	M	98	3\6	10%	B+ve	Nil	40	4	7800	245	3	106	88	186	256	N	Nil	Nil	AR Pulsed	Nil	AR
68	Hemavathy	45	F	CIN	0	A+	50	F	S	102	ND	10%	A+ve	Nil	34	5	8600	196	3	88	86	90	98	N	Nil	LYM	Nil	cellulitis	ND
69	Perumal	43	M	NK	0	O+	48	F	S	98	ND	10%	O+ve	PRESENT	55	4	3400	342	8	124	116	140	188	SGF	Nil	Nil	Nil	Nil	ND
70	Ali Baba	23	M	CGN	0	AB+	45	F	M	90	3\6	5%	B+ve	Nil	40	3	8900	230	4	98	90	88	92	N	Nil	Nl	Nil	Nil	ND
71	Sankar	28	M	NK	0	B+ve	50	F	M	106	3\6	10%	O+ve	nil	45	3	10800	105	3	123	281	260	306	N	Pos	nil	GDF	nil	ND
72	Velu	18	M	CGN	0	O+	47	F	M	96	3\6	5%	O+ve	nil	40	2	5150	150	5	90	103	122	124	N	Nil	nil	Nil	nil	ND
73	Santhanam	20	M	NIK	0	B+	48	F	M	89	ND	5%	O+ve	nil	35	3	7800	214	5	184	140	108	110	N	Nil	nil	Nil	Pneum	ND
74	Kumar	25	M	CGN	0	A1-ve	45	F	M	93	3\6	15%	A1-ve	nil	45	3	10600	155	6	90	103	99	101	N	Nil	nil	Nil	nil	ND
75	Suseela	30	F	NK	0	B+	38	M	B	116	3\6	5%	O+ve	Nil	40	3	9600	220	3	98	88	90	90	N	Nil	Nil	Nil	Nil	ND
76	mani	23	M	IgAN	0	AB+	45	F	M	98	3\6	5%	B+ve	nil	40	5	11800	214	4	90	98	88	88	N	Nil	Nil	Nil	UTI	ND
77	Raja	25	M	IGAN	0	O+ve	46	F	M	89	5\6	5%	O+ve	nil	40	3	12300	193	4	123	136	138	387	N	Nil	nil	AR Pulsed	nil	AR
78	Raghu	22	M	CGN	0	B+ve	38	F	M	89	3\6	5%	O+ve	nil	40	3	8400	123	3	96	88	100	86	N	Nil	nil	Nil	nil	ND
79	Shanmugam	30	M	CGN	0	B+ve	40	F	S	104	3\6	10%	O+ve	nil	40	3	9850	198	4	105	124	154	134	N	Pos	nil	GDF	nil	ND